Spontaneous Renal Artery Dissection

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Spontaneous renal artery dissection (SRAD) is a rare event, and thus may be a challenge for physicians to diagnose and treat. We report a case of SRAD in a healthy 56-year-old male who presented with flank pain, fever, and elevated white blood cell count. The patient was initially diagnosed with nephrolithiasis versus pyelonephritis and was admitted for observation. Multiple imaging modalities, including non-contrast computed tomography (CT), magnetic resonance imaging (MRI) with gadolinium, CT angiogram, and intraoperative angiogram, were used to make the final diagnosis of SRAD. The patient was treated with endovascular stent placement and is currently free of pain with normal laboratory values and blood pressure.
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Spontaneous renal artery dissection (SRAD) is a rare occurrence with fewer than 200 cases reported in the literature. Due to its rarity, SRAD may be difficult to diagnose and treat. A number of imaging modalities, such as computed tomography (CT) scan, intravenous pyelogram (IVP), or magnetic resonance imaging (MRI) may be useful in diagnosis, but the gold standard is angiogram. Treatment options vary depending on the severity of the patient's clinical condition. Observation with anticoagulation, endovascular procedures, open vascular surgery, and nephrectomy have all been effective treatment modalities. This case report describes a healthy gentleman who presented with flank

pain, fever, and elevated white blood cell count, and was diagnosed with SRAD after undergoing a number of imaging tests, including angiogram. We discuss the diagnostic work up and treatment plan associated with this particular patient and SRAD in general.

Case Presentation

A healthy 56-year-old male presented to our emergency room with complaints of right-sided flank pain and fever for approximately 3 days. He stated his pain began suddenly while on a business trip out of state. At that time, he went to the local ER, where an intravenous pyelogram (IVP) was performed. The patient was discharged with pain medications and was medically cleared to fly home later that day.

On the day of presentation to our facility, he complained of nausea for 2 days and decreased oral intake. He denied gross hematuria and voiding symptoms but was febrile to 103°F with chills at home. He brought the IVP report from the outside ER, which described a scout film without calcifications and a delayed nephrogram on the right through 1 hour and 20 minutes. By report, there was no evidence of excretion from the right kidney, making this consistent with "high grade obstruction of the right kidney."

The patient denied past medical history, surgical history, and allergies, but had a history of smoking 30-pack years. He had a low-grade temperature of 100.3°F in the ER with a heart rate of 122 bpm and normal blood pressure. On physical examination, he was alert and oriented with slight facial flushing. He was tachycardic with a regular rhythm and clear breath sounds. His abdomen was soft, nontender, and non-distended with normal bowel sounds, and he had no costovertebral tenderness bilaterally. His laboratory values were significant for WBC 14.4 and Hct 44.7. His BUN and Cr were 12 and 1.4 respectively. His liver enzymes were slightly elevated with an ALT of 225 and AST of 296. His bilirubins were within normal limits. His urinalysis showed small blood, negative nitrite, and trace leukocyte esterase activity. Microscopic evaluation revealed 11-25 RBCs and 6-10 WBCs with few bacteria.

A non-contrast CT scan of the abdomen and pelvis was performed in the emergency room to evaluate for kidney stones (Figure 1). A 2-3 mm non-obstructing stone was seen in the lower pole of the left kidney without hydronephrosis. Perinephric stranding was present bilaterally, right greater than left, allowing the possible diagnosis of previously passed right-sided renal calculus. An abdominal ultrasound performed to further evaluate the elevated liver function tests (LFTs) showed no evidence of gallstones or gall bladder wall thickening.

The patient was admitted to the urology service for fever and right flank pain with a presumed diagnosis of passed kidney stone versus pyelonephritis. His elevated LFTs were attributed to dehydration, and he was started on IV fluids as well as empiric IV ampicillin and gentamicin. The following day his pain was improved, and he was tolerating a regular diet. He remained febrile to 102.3°F with a white blood cell count of 13.1 and Cr of 1.2. His LFTs began to normalize to AST/ALT 76/122. A gastroenterology consult recommended magnetic resonance cholangiopancreatography (MRCP) to evaluate for a retained bile duct stone. On hospital day 2 the patient continued to be febrile (101.6°F) with stable vital signs. His labs included WBC 12.2, Cr 1.1, and AST/ALT 38/113. The MRCP showed a normal biliary system, but revealed right renal cortical and medullary wedge-shaped areas of hypoperfusion with enhancement of the capsule (Figure 2). This was thought to be most consistent with renal infarction. The right renal artery was patent, but there was presence of a high signal on T1-weighted sequences, possibly representing dissection. A CT-angiogram was recommended by the radiologist to further assess the artery. These images (Figure 3) revealed multiple peripheral infarcts in the right kidney with a small amount of fluid/edema in the right perinephric space, which was slightly increased from the previous non-contrast CT. In addition, there was a linear filling defect of the right renal artery consistent with dissection and intramural hematoma. A vascular surgery consult was obtained and recommended an angiogram, which was performed the following day (Figure 4). At the time of angiogram, a right renal artery stent was placed. The patient was discharged home the next day afebrile on Plavix. His creatinine and liver enzymes were within normal limits, and he was given appointments to follow up with both his urologist and the vascular surgeon in 1 month.

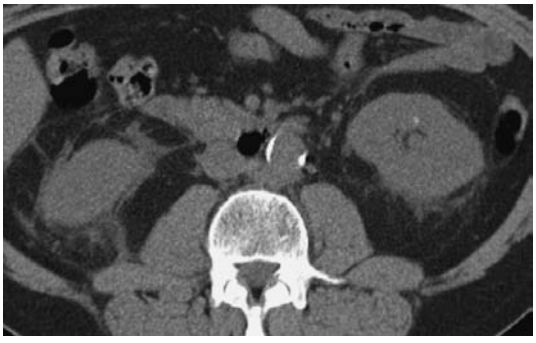


Figure 1. *Non-contrast computed tomography scan showing a small non-obstructing stone in the LLP and perinephric stranding of both kidneys, right greater than left.*

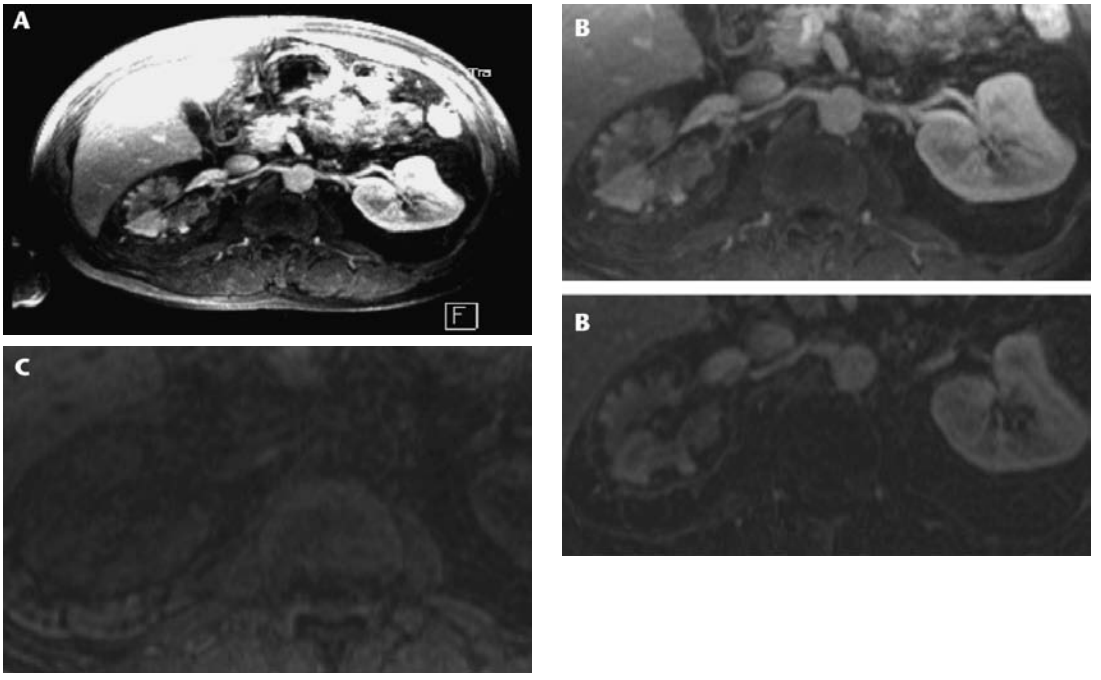
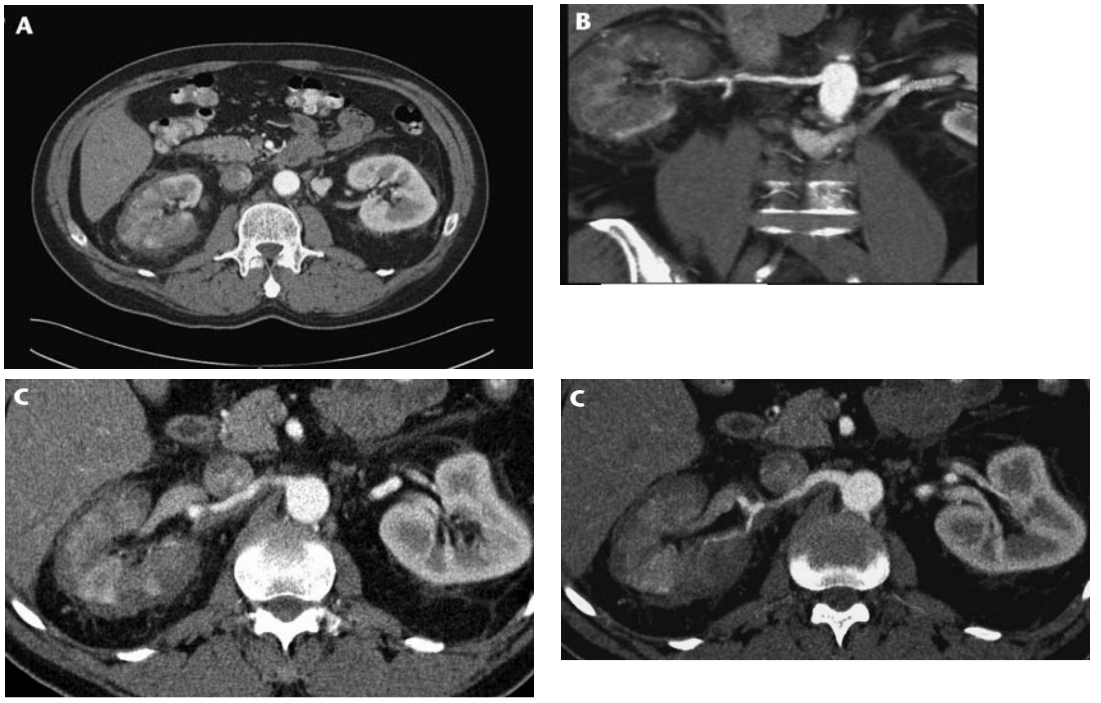


Figure 2. (A) *Magnetic resonance cholangiopancreatography (MRCP).* **(B)** *T1 post contrast MRCP showing right renal cortical and medullary wedge-shaped areas of hypoperfusion with enhancement of the capsule, thought to be most consistent with renal infarction.* **(C)** *T1 pre-contrast MRCP showing patent right renal artery with high signal intensity posterior in the artery, possibly representing dissection.*

Figure 3. (A) *Computed tomography (CT) with contrast revealing multiple peripheral infarcts in the right kidney with a small amount of fluid/edema in the right perinephric space, slightly increased from previous non-contrast CT.* **(B)** *CT angiogram showing a linear filling defect of the right renal artery consistent with dissection and intramural hematoma.* **(C)** *CT angiogram once again showing the linear filling defect in the right renal artery, consistent with dissection and intramural hematoma.*



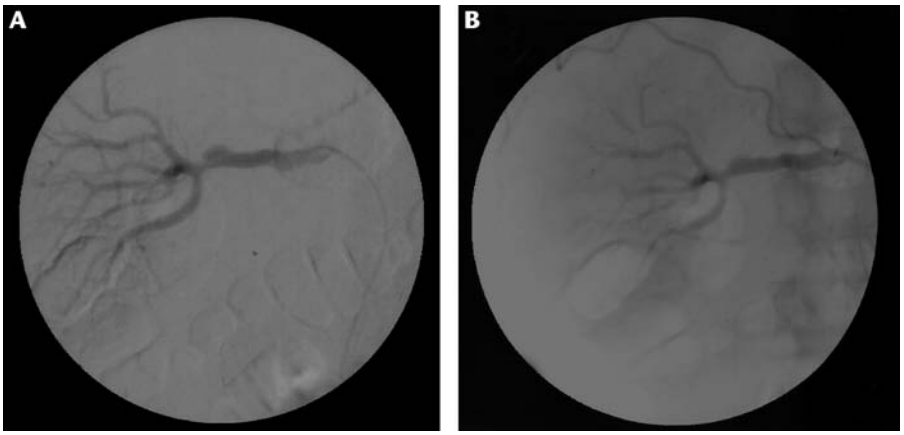


Figure 4. (A) *Intra-operative angiogram showing right renal artery dissection prior to stent placement.* (B) *Intra-operative angiogram after placement of a stent in the right renal artery.*

Discussion

Renal artery dissection rarely occurs as an isolated, non-traumatic event. In almost all cases the etiology is certain, whether from the natural extension of aortic dissection, as a consequence of percutaneous angioplasty, or as a result of blunt abdominal trauma.[1,2] Due to its rarity, SRAD often presents as a diagnostic and therapeutic challenge.

Almost 200 cases of spontaneous renal artery dissection have been published in the literature.[2] Although the reported incidence is low, it is probably a more common phenomenon because many may be silent or resolve spontaneously. SRAD is usually observed in otherwise healthy men in the fourth to sixth decade of life and occurs in a 4:1 male to female ratio.[1-3] Bilateral lesions have been seen in 10%-15% of cases, and there is no evidence that one kidney is more susceptible than the other.[1,4-6] Conditions associated with the development of SRAD include fibromuscular dysplasia, malignant hypertension, severe atherosclerosis, Marfan syndrome, Ehlers-Danlos syndrome, subadventitial angioma, cystic medial necrosis, and extreme physical exertion.[1,2,4,7,8] In the majority of cases, however, the cause of SRAD is never found.

Clinical manifestations of SRAD include progressive renovascular hypertension, changes in renal function, and symptoms of kidney infarction.[1] The initial presentation can be nonspecific with symptoms suggestive of renal colic, as in our scenario. The most common symptom in acute dissection is severe pain occurring unilaterally in the upper abdomen or flank with radiation to the epigastrium.[1] Hypertension occurs in almost all patients. Because this condition is rare and the symptoms are vague, the diagnosis is often not considered until additional imaging studies are performed.[1,2] CT often shows areas of infarction, suggesting a vascular etiology. Angiography is the most useful test, because it can precisely demonstrate the extent and nature of the vascular involvement while identifying potential treatment options.[1] Angiography also serves as a baseline study to be used in comparison with follow-up examinations.[1]

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The natural history of SRAD is poorly understood due to the infrequency of cases and the lack of patient follow-up. The major long-term effect is malignant hypertension, and mortality generally results from renal failure secondary to ischemia, although more commonly with bilateral dissections.[1,2,9]

There are a number of treatment options for SRAD, and the physician's approach should be based on stability of the lesion and renal function. For example, if serial angiography shows stability of a lesion with no deterioration of renal function, careful follow-up may be feasible.[1] On the other hand, some form of intervention may be required if repeat angiography shows an unstable lesion or if hypertension remains uncontrollable with maximal medical therapy.[1,2] In addition, acute deterioration of renal function necessitates intervention.[1,2] Management options include observation with anticoagulation,[2,10,11] surgical therapy such as vascular reconstruction[4,5,10,12,13] or nephrectomy, and endovascular procedures such as stenting[14] or coiling.[1] Anticoagulation seems to produce satisfactory short-term results,[2,10,11] but long-term results are lacking. Surgical management of SRAD is intended to treat renovascular hypertension and to preserve kidney function. Surgical revascularization is indicated in kidneys with substantial residual renal function, while primary nephrectomy should be considered if the kidney is already severely damaged due to infarction, has poor function on isotope renography, or if revascularization would be difficult because of renal branch artery involvement.[10,11]

In this scenario, we opted to treat the dissection with endovascular management in conjunction with the vascular surgery team. Various case reports in the literature demonstrate successful return of normal renal blood flow and controlled hypertension after stenting.[14-16] Our patient was free of pain after stenting, with normalized laboratory values and blood pressure. The most recent imaging performed in follow-up shows preservation of flow in the cortex of the right lower pole with loss of cortical tissue in the superior aspect of the right kidney, consistent with infarction. The patient continues to have a normal blood pressure without antihypertensive therapy. We will continue to follow his progress along with the vascular surgeons.

In summary, isolated SRAD is rare and often presents as a diagnostic and therapeutic challenge. Advanced imaging modalities are helpful in making the diagnosis, but angiography remains the definitive study. The treatment and long-term management of patients with this condition are poorly understood and controversial, and a commitment to long-term follow-up is often required from both the patient and the physicians involved. ■

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Main Points

- Spontaneous renal artery dissection is a rare condition that is usually diagnosed after imaging studies are performed for abdominal or flank pain.
- Angiography is the definitive test, as it precisely demonstrates the extent of vascular involvement, identifies treatment options, and provides a baseline study for comparison with future studies.
- Expectant management with anticoagulation may produce satisfactory short-term results.
- Revascularization with surgical or endovascular techniques should be reserved for patients with uncontrollable hypertension, renal insufficiency, or bilateral dissections with compromise of both renal arteries.
- Nephrectomy may be of benefit in cases where revascularization is difficult or not possible.